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Limits: Publication Date to 2003/10/3

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Search	Most Recent Queries	Time	Result
#19	Search myosin-9 and antibody and tumor Limits: Publication Date to 2003/10/3	12:01:30	21
#17	Search myosin-9 and antibody Limits: Publication Date to 2003/10/3	12:00:53	275
#12	Search myosin-9 Limits: Publication Date to 2003/10/3	12:00:45	2458
#15	Search nmmhc-a Limits: Publication Date to 2003/10/3	11:59:16	5
#13	Search myosin-9 and (cancer or tumor or tumour or carcinoma) and antibody Limits: Publication Date to 2003/10/3	11:58:14	21
#9	Search (myosin heavy chain type A) and antibody and (cancer or tumor or tumour or carcinoma) Limits: Publication Date to 2003/10/3	11:49:22	11
#8	Search (myosin heavy chain type A) and antibody Limits: Publication Date to 2003/10/3	11:49:03	230
#7	Search (myosin heavy chain type A) Limits: Publication Date to 2003/10/3	11:48:56	1646
#3	Search (myosin heavy chain type A) and (cancer or tumor) and antibody Limits: Publication Date to 2003/10/3	11:44:34	11
#2	Search (myosin heavy chain type A) and (cancer or tumor) Limits: Publication Date to 2003/10/3	11:44:20	62
#1	Search myosin heavy chain type A and cancer or tumor Limits: Publication Date to 2003/10/3	11:43:52	1731262

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Dec 18 2006 06:34:27

WEST Search History

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DATE: Thursday, December 21, 2006

<u>Hide?</u>	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L8	myosin 9	0
<input type="checkbox"/>	L7	myosin-9	0
<input type="checkbox"/>	L6	(myosin 9) and antibody	0
<input type="checkbox"/>	L5	myosin-9 and antibody	0
<input type="checkbox"/>	L4	(myosin heavy chain type a) and antibody	9
<input type="checkbox"/>	L3	(myosin heavy chain type a) and antibody and (cancer or tumor or tumour or carcinoma)	9
<input type="checkbox"/>	L2	nmmhc-a and antibody and (cancer or tumor or tumour or carcinoma)	9
<input type="checkbox"/>	L1	myosin-9 and antibody and (cancer or tumor or tumour or carcinoma)	0

END OF SEARCH HISTORY

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Last logoff: 11dec06 12:33:22
Logon file001 21dec06 13:15:12

***** ANNOUNCEMENTS *****

NEW FILES RELEASED

***Engineering Index Backfile (File 988)
***Verdict Market Research (File 769)
***EMCare (File 45)
***Trademarkscan - South Korea (File 655)

RESUMED UPDATING

***File 141, Reader's Guide Abstracts

RELOADS COMPLETED

***Files 340, 341 & 942, CLAIMS/U.S. Patents - 2006 reload now online
***Files 173 & 973, Adis Clinical Trials Insight
***File 11, PsycInfo
***File 531, American Business Directory

DATABASES REMOVED

***File 196, FINDEX

***File 468, Public Opinion Online (POLL)

Chemical Structure Searching now available in Prous Science Drug

Data Report (F452), Prous Science Drugs of the Future (F453), IMS R&D Focus (F445/95
Facts (F390), Derwent Chemistry Resource (F355) and Index Chemicus
(File 302).

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>>>a specific database by entering HELP NEWS <file number>. <<<

* * *

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Accession numbers have changed.

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B 155, 159, 10, 203, 35, 5, 467, 73, 434, 34
21dec06 13:15:52 User290558 Session D88.1
\$0.93 0.265 DialUnits File1
\$0.93 Estimated cost File1
\$0.18 INTERNET
\$1.11 Estimated cost this search
\$1.11 Estimated total session cost 0.265 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 155: MEDLINE(R) 1950-2006/Dec 06
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***File 155: MEDLINE has temporarily stopped updating with UD=20061206.**
Please see HELP NEWS154 for details.

File 159:Cancerlit 1975-2002/Oct
(c) format only 2002 Dialog

*File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.

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File 34:SciSearch(R) Cited Ref Sci 1990-2006/Dec W3
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Set Items Description

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?

S MYOSIN (N) 9
143508 MYOSIN
4057348 9
S1 62 MYOSIN (N) 9

?

S S1 AND ANTIBODY
62 S1
1794137 ANTIBODY
S2 1 S1 AND ANTIBODY

?

TYPE S2/FULL/1

2/9/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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07072077 PMID: 2942552

Isolation and partial characterization of a 110-kD dimer actin-binding protein.

Ueno T; Korn E D
Journal of cell biology (UNITED STATES) Aug 1986, 103 (2) p621-30,
ISSN 0021-9525--Print Journal Code: 0375356

Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

Two Triton-insoluble fractions were isolated from Acanthamoeba castellanii. The major non-membrane proteins in both fractions were actin (30-40%), myosin II (4-9%), myosin I (1-5%), and a 55-kD polypeptide (10%). The 55-kD polypeptide did not react with antibodies against tubulins from turkey brain, paramecium, or yeast. All of these proteins were much more concentrated in the Triton-insoluble fractions than in the whole homogenate or soluble supernatant. The 55-kD polypeptide was extracted with 0.3 M

NaCl, fractionated by ammonium sulfate, and purified to near homogeneity by DEAE-cellulose and hydroxyapatite chromatography. The purified protein had a molecular mass of 110 kD and appeared to be a homodimer by isoelectric focusing. The 110-kD dimer bound to F-actin with a maximal binding stoichiometry of 0.5 mol/mol of actin (1 mol of 55-kD subunit/mol of actin). Although the 110-kD protein enhanced the sedimentation of F-actin, it did not affect the low shear viscosity of F-actin solutions nor was bundling of F-actin observed by electron microscopy. The 110-kD dimer protein inhibited the actin-activated Mg²⁺-ATPase activities of Acanthamoeba myosin I and myosin II in a concentration-dependent manner. By indirect immunofluorescence, the 110-kD protein was found to be localized in the peripheral cytoplasm near the plasma membrane which is also enriched in F-actin filaments and myosin I.

Descriptors: *Amoeba--analysis--AN; *Carrier Proteins --isolation and purification--IP; *Cytoskeletal Proteins--isolation and purification--IP; *Microfilament Proteins; Adenosinetriphosphatase--metabolism--ME; Cell Compartmentation; Fluorescent Antibody Technique; Gelsolin; Magnesium --metabolism--ME; Molecular Weight; Polyethylene Glycols; Research Support, Non-U.S. Gov't; Solubility

CAS Registry No.: 0 (Carrier Proteins); 0 (Cytoskeletal Proteins); 0 (Gelsolin); 0 (Microfilament Proteins); 0 (Polyethylene Glycols); 0 (brevin); 7439-95-4 (Magnesium)

Enzyme No.: EC 3.6.1.3 (Adenosinetriphosphatase)

Record Date Created: 19860917

Record Date Completed: 19860917

?

Set	Items	Description
S1	62	MYOSIN (N) 9
S2	1	S1 AND ANTIBODY

S (NMMHC (W) A) AND ANTIBODY
107 NMMHC
42195946 A
26 NMMHC (W) A
1794137 ANTIBODY
S3 1 (NMMHC (W) A) AND ANTIBODY

?

TYPE S3/FULL/1

3/9/1 (Item 1 from file: 155)
DIALOG(R)File 155: MEDLINE(R)
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14454941 PMID: 12930685

[Immunofluorescence localization of inclusion and identification of nonmuscle myosin heavy chain IIA in neutrophils of May-Hegglin anomaly patients]

Yi Yan; Zhang Guang-sen
Department of Hematology, Institute of Molecular Hematology, Second Xiangya Hospital, Central South University, Changsha 410011, China.
Zhonghua yi xue za zhi (China) Aug 10 2003, 83 (15) p1313-6, ISSN 0376-2491--Print Journal Code: 7511141
Publishing Model Print
Document type: Journal Article ; English Abstract
Languages: CHINESE
Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

OBJECTIVE: To observe the localization of inclusion and expression of nonmuscle myosin heavy chain-A (NMMHC-A) in cytoplasm of neutrophils of May-Hegglin anomaly (MHA) patients, and elucidate and identify the property of the inclusions in constitutional elements. METHODS: Peripheral blood was drawn from the MHA proband, the proband's father, and a healthy control. White blood cells and platelets were isolated and smeared. Indirect immunofluorescence technique combined with propidium iodide (PI) nuclei staining technology was used to detect the inclusion and nonmuscle myosin in cytoplasm of neutrophils and platelet. Neutrophils were isolated. Protein in the neutrophils was extracted and underwent Western blot assay to examine the expression of NMMHC-A. RESULTS: Spindle-like inclusions with yellow fluorescence were clearly displayed in the neutrophils of the MHA patient and her father, that matched very well in shape, size and localization with the inclusions, revealed by Wright-Giemsa's stain. In normal control, except a diffusive distribution of fluorescent spot in neutrophils cytoplasm, not any inclusion was detected. As for NMMHC-A expression, Western blot assay showed that NMMHC-A was upregulated in the neutrophils of the MHA patient (60.9) and her father (58.9). CONCLUSION: A new method to display MHA inclusions and identify the major component of inclusions in the neutrophils, which was originated from a mutant of nonmuscle myosin, of MHA was set up. Immunofluorescence analysis is more sensitive than Wright-Giemsa's staining in detecting inclusions of MHA.

Tags: Female; Male

Descriptors: *Inclusion Bodies--ultrastructure--UL; *Myosin Heavy Chains --blood--BL; *Neutrophils--chemistry--CH; *Thrombocytopenia--genetics--GE; Chromosomes, Human, Pair 22; English Abstract; Fluorescent Antibody Technique, Indirect; Humans; Molecular Motors; Research Support, Non-U.S. Gov't; Syndrome; Thrombocytopenia--blood--BL; Thrombocytopenia--pathology --PA

CAS Registry No.: 0 (MYH9 protein, human); 0 (Molecular Motors); 0 (Myosin Heavy Chains)

Record Date Created: 20030821

Record Date Completed: 20040204

?

Set	Items	Description
S1	62	MYOSIN (N) 9
S2	1	S1 AND ANTIBODY
S3	1	(NMMHC (W) A) AND ANTIBODY

?

S (NMMHC (W) A) AND (CANCER OR TUMOR OR TUMUOR OR CARCINOMA OR NEOPLASIA)
107 NMMHC
42195946 A
26 NMMHC(W)A
3585741 CANCER
3338516 TUMOR
56 TUMUOR
1782646 CARCINOMA
141730 NEOPLASIA
S4 0 (NMMHC (W) A) AND (CANCER OR TUMOR OR TUMUOR OR CARCINOMA OR NEOPLASIA)

?

S (NMMHC (W) A)
107 NMMHC
42195946 A

S5 26 (NMMHC (W) A)
?

S (NMMHC (W) A)
107 NMMHC
42195946 A
S6 26 (NMMHC (W) A)
?

S S6 AND ANTIBODY
26 S6
1794137 ANTIBODY
S7 1 S6 AND ANTIBODY
?

TYPE S7/FUL/1
>>>"FUL" is not a valid format name in file(s): 5, 10, 34-35, 73, 155, 159,
203, 434, 467
?

Set	Items	Description
S1	62	MYOSIN (N) 9
S2	1	S1 AND ANTIBODY
S3	1	(NMMHC (W) A) AND ANTIBODY
S4	0	(NMMHC (W) A) AND (CANCER OR TUMOR OR TUMUOR OR CARCINOMA - OR NEOPLASIA)
S5	26	(NMMHC (W) A)
S6	26	(NMMHC (W) A)
S7	1	S6 AND ANTIBODY

?

TYPE S7/FULL/1

7/9/1 (Item 1 from file: 155)
DIALOG(R)File 155: MEDLINE(R)
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14454941 PMID: 12930685
[Immunofluorescence localization of inclusion and identification of
nonmuscle myosin heavy chain IIA in neutrophils of May-Hegglin anomaly
patients]
Yi Yan; Zhang Guang-sen
Department of Hematology, Institute of Molecular Hematology, Second
Xiangya Hospital, Central South University, Changsha 410011, China.
Zhonghua yi xue za zhi (China) Aug 10 2003, 83 (15) p1313-6, ISSN
0376-2491--Print Journal Code: 7511141
Publishing Model Print
Document type: Journal Article ; English Abstract
Languages: CHINESE
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
OBJECTIVE: To observe the localization of inclusion and expression of
nonmuscle myosin heavy chain-A (NMMHC-A) in cytoplasm of neutrophils of
May-Hegglin anomaly (MHA) patients, and elucidate and identify the property
of the inclusions in constitutional elements. METHODS: Peripheral blood was
drawn from the MHA proband, the proband's father, and a healthy control.
White blood cells and platelets were isolated and smeared. Indirect
immunofluorescence technique combined with propidium iodide (PI) nuclei

staining technology was used to detect the inclusion and nonmuscle myosin in cytoplasm of neutrophils and platelet. Neutrophils were isolated. Protein in the neutrophils was extracted and underwent Western blot assay to examine the expression of NMMHC-A. RESULTS: Spindle-like inclusions with yellow fluorescence were clearly displayed in the neutrophils of the MHA patient and her father, that matched very well in shape, size and localization with the inclusions, revealed by Wright-Giemsa's stain. In normal control, except a diffusive distribution of fluorescent spot in neutrophils cytoplasm, not any inclusion was detected. As for NMMHC-A expression, Western blot assay showed that NMMHC-A was upregulated in the neutrophils of the MHA patient (60.9) and her father (58.9). CONCLUSION: A new method to display MHA inclusions and identify the major component of inclusions in the neutrophils, which was originated from a mutant of nonmuscle myosin, of MHA was set up. Immunofluorescence analysis is more sensitive than Wright-Giemsa's staining in detecting inclusions of MHA.

Tags: Female; Male

Descriptors: *Inclusion Bodies--ultrastructure--UL; *Myosin Heavy Chains --blood--BL; *Neutrophils--chemistry--CH; *Thrombocytopenia--genetics--GE; Chromosomes, Human, Pair 22; English Abstract; Fluorescent Antibody Technique, Indirect; Humans; Molecular Motors; Research Support, Non-U.S. Gov't; Syndrome; Thrombocytopenia--blood--BL; Thrombocytopenia--pathology --PA

CAS Registry No.: 0 (MYH9 protein, human); 0 (Molecular Motors); 0 (Myosin Heavy Chains)

Record Date Created: 20030821

Record Date Completed: 20040204

?

Set	Items	Description
S1	62	MYOSIN (N) 9
S2	1	S1 AND ANTIBODY
S3	1	(NMMHC (W) A) AND ANTIBODY
S4	0	(NMMHC (W) A) AND (CANCER OR TUMOR OR TUMUOR OR CARCINOMA - OR NEOPLASIA)
S5	26	(NMMHC (W) A)
S6	26	(NMMHC (W) A)
S7	1	S6 AND ANTIBODY

?

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 GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
 LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ,
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 TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
 KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
 FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
 BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
 CA 2501222 A1 20041021 CA 2003-2501222 20031003
 AU 2003271093 A1 20041101 AU 2003-271093 20031003
 EP 1559725 A1 20050803 EP 2003-751332 20031003
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
 US 2006057147 A1 20060316 US 2005-530171 20050517
 PRAI JP 2002-291953 A 20021004
 WO 2003-JP12732 W 20031003
 AB Provided are ligands and antibodies specific to cell surface antigen of solid tumor such as myosin, especially non-muscle type myosin heavy chain type A. These ligands and monoclonal antibodies are conjugated or labeled with antitumor agent, antitumor protein, enzyme, gene or radioisotope for diagnosis and treatment of cancer such as stomach cancer, breast cancer, colon cancer or esophageal cancer.
 RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> s (myosin 9)
L4      4 (MYOSIN 9)

=> s 14 and antibody
L5      1 L4 AND ANTIBODY

=> d 15 bib abs 1
  
```

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
 AN 2002:937303 CAPLUS
 DN 138:20443
 TI Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
 IN Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
 PA Takara Bio Inc., Japan
 SO Jpn. Kokai Tokkyo Koho, 386 pp.
 CODEN: JKXXAF

DT Patent
 LA Japanese
 FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2002355079	A	20021210	JP 2002-69354	20020313
PRAI JP 2001-73183	A	20010314		
JP 2001-74993	A	20010315		
JP 2001-102519	A	20010330		

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises preparing a nucleic acid sample containing mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample containing the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is

altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-β estradiol (E2), were found in mice by DNA chip anal.

=> s myosin and antibody and (cancer or tumor or tumuor or carcinoma or malignancy)
L6 552 MYOSIN AND ANTIBODY AND (CANCER OR TUMOR OR TUMUOR OR CARCINOMA
OR MALIGNANCY)

=> s 16 and (non muscle myosin heavy chain)
L7 4 L6 AND (NON MUSCLE MYOSIN HEAVY CHAIN)

=> duplicate remove 17
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, BIOTECHDS, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L7
L8 2 DUPLICATE REMOVE L7 (2 DUPLICATES REMOVED)

=> d 18 bib abs 1-2

L8 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
AN 2004-14428 BIOTECHDS
TI Identifying a compound that modulates angiogenesis or tumorigenesis, useful in diagnosing and treating angiogenesis, cancer, stroke, infertility and heart disease, comprises contacting the compound with angiogenesis polypeptide; antisense molecule and RNA interference for use in disease therapy and gene therapy
AU LORENS J B; ATCHISON R E; FRIERA A; HOLLAND S
PA RIGEL PHARM INC
PI WO 2004039955 13 May 2004
AI WO 2003-US34281 29 Oct 2003
PRAI US 2003-512251 17 Oct 2003; US 2002-421989 29 Oct 2002
DT Patent
LA English
OS WPI: 2004-376181 [35]
AN 2004-14428 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Identifying a compound that modulates angiogenesis or tumorigenesis comprises contacting the compound with angiogenesis polypeptide.
DETAILED DESCRIPTION - Identifying a compound that modulates angiogenesis or tumorigenesis comprises: (a) contacting the compound with angiogenesis polypeptide, e.g. Ax1, tubulin cofactor D, transglutaminase 2, cytosine deaminase, peptidase M41 (paraplegin), CD13 aminopeptidase, PPK-1, zip kinase, Gas6, SRm160, non-muscle myosin heavy chain, calmodulin 2, novel symporter, novel semaphorin, novel zinc finger helicase (FLJ22611), plexin-A2, deoxycytidylate deaminase or novel sugar transporter; (b) determining the functional effector of the compound upon the angiogenesis polypeptide or the physical effect of the compound upon the target polypeptide or its fragment or inactive variant; and (c) determining the chemical or phenotypic effect of the compound upon a cell comprising the target polypeptide or its fragment or inactive variant, thus identifying a compound that modulates cell cycle arrest. An INDEPENDENT CLAIM is also included for a method of modulating angiogenesis in a subject.

BIOTECHNOLOGY - Preferred Method: Specifically, identifying a compound that modulates tumorigenesis comprises contacting the compound with an Ax1 polypeptide, determining the functional or physical effect of the compound upon the Ax1 polypeptide or its fragment or inactive variant and determining the chemical or phenotypic effect of the compound upon a cell comprising the Ax1 polypeptide or its fragment or inactive variant. The functional effect is determined in vitro. The functional effect is a

physical effect. The functional effect is determined by measuring ligand binding to the polypeptide. The functional effect is a chemical or phenotypic effect. The polypeptide is expressed in a eukaryotic host cell. The host cell is an endothelial cell. The functional effect is determined by measuring alphavbeta3 expression or haptotaxis. Modulation is inhibition of angiogenesis or tumorigenesis. The polypeptide is recombinant. The compound is an antibody, an antisense molecule, an RNAi molecule or a small organic molecule. The host cell is a cancer cell. Modulating angiogenesis in a subject comprises administering to the subject a therapeutical amount of the compound identified by the method above. The subject is human. The compound inhibits angiogenesis or tumorigenesis.

ACTIVITY - Antiangiogenic; Cytostatic; Cerebroprotective; Vasotropic; Antiinfertility; Cardiant. No biological data given.

MECHANISM OF ACTION - Antibody Therapy; Antisense Therapy; RNAi Therapy.

USE - The method is useful in identifying a compound that modulates angiogenesis. The methods and compounds or compositions are useful in diagnosing and treating angiogenesis, cancer, stroke, infertility and heart disease.

ADMINISTRATION - Dosage is 1-100 mug/70 kg by injection, oral, inhalation, transdermaal, rectal or parenteral (e.g. intraarticular, intravenous, intramuscular, intradermal, intraperitoneal or subcutaneous) means.

EXAMPLE - No relevant example given. (105 pages)

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 1994:531214 CAPLUS
DN 121:131214
TI Non-muscle myosin heavy chain as a possible target for protein encoded by metastasis-related mts-1 gene
AU Krajewska, Marina V.; Cardenas, Mauricio Neira; Grigorian, Mariam S.; Ambartsumian, Noona S.; Georgiev, Georgii P.; Lukyanidin, Eugene M.
CS Dep. Mol. Cancer Biol., Danish Cancer Soc., Copenhagen, DK-2100, Den.
SO Journal of Biological Chemistry (1994), 269(31), 19679-82
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The mts-1 gene is associated with the expression of the metastatic phenotype of tumor cells. The protein product of the mts-1 gene belongs to the S100 family of Ca²⁺-binding proteins with unknown biochem. function. In the present work, monoclonal anti-Mts-1 antibodies were used to isolate and characterize Mts-1 protein possible targets. Mts-1 protein can be immunopptd. by both anti-Mts-1 and anti-myosin antibodies as a complex with myosin from lysates of different mouse and human cell lines. Precipitation of myosin by anti-Mts-1 antibodies is specific and depends on the presence of Mts-1 protein. Ca²⁺-dependent association between Mts-1 protein and the heavy chain of non-muscle myosin was demonstrated by blot overlay technique. Furthermore, association between myosin and Mts-1 was confirmed by sucrose gradient anal. Finally, immunofluorescent staining of the mouse mammary adenocarcinoma cell line showed that Mts-1 protein is co-localized with the myosin complex. The data suggest that the target for Mts-1 protein is a heavy chain of non-muscle myosin.

=> s (non muscular myosin heavy chain)
L9 2 (NON MUSCULAR MYOSIN HEAVY CHAIN)

=> s 19 and antibody
L10 0 L9 AND ANTIBODY